pain is still present and the dog continues to carry its leg, amputation of the femoral head is recommended. This results in an immediate and dramatic freedom from pain. Whether an arthroplasty is performed or a femoral head prosthesis fitted is a matter of choice but in small dogs the former procedure leaves little to be desired.

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Arthropathy in Pigs

The principal bacteria associated with arthritis in pigs are *Erysipelothrix insidiosa*, streptococci and hæmophilus. The course of erysipelas is well documented (Collins & Goldie 1940). Vaccination is widely practised and protects against the acute form of the disease, although it may sensitize the pig to the chronic, arthritic form (Freeman & Berman 1964). As *E. insidiosa* is almost ubiquitous, and as arthritis due to hypersensitivity to it can occur without organisms being demonstrable in the lesions, it is possible to postulate that most arthropathy in pigs stems from the organism (Duthie & Lancaster 1964). Grabell *et al.* (1962) have made a study of discospondylitis due to erysipelothrix.

More spectacular arthritis resembling Glässers disease has been described associated with a PPLO and hæmophilus (Lecce 1960) and with *Mycoplasma hyorhinis* (Roberts *et al.* 1963). Classical joint ill and navel ill associated with pyogenic bacteria also occur in pigs.

I do not believe that all arthropathy in pigs arises from an infectious cause. Sabec (1960) has described a tarsitis of pigs, which probably has a non-infectious ætiology, and it is probable that a large proportion of other joint lesions occur in the absence of infectious agents. It is, in fact, quite difficult to find a pig over the weight of 75 kg that does not have some arthritic condition. To understand this it is necessary to know the background to modern pig husbandry.

Few pigs live to a ripe old age, so that senile changes are not a problem. Some 95% of all pigs reared in this country are killed at either 4 or 7 months for the pork or bacon markets. They are, of course, kept for profit, and variations in production cost of a penny per pound live weight can halve this profit. A higher price is paid for pigs which produce the sort of joints that the bacon factory prefers – the so-called 'desirable carcase characteristics'. In intensive units it is also preferable that the pigs should grow rapidly so that

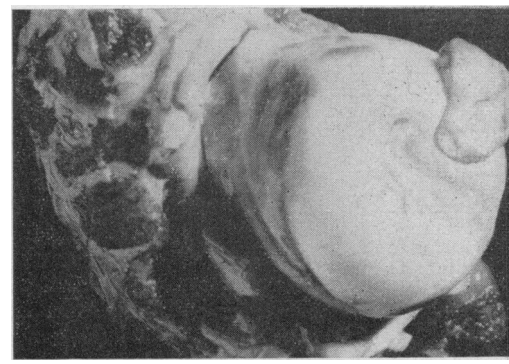

Fig 1 *Wear lesions in the femoral head*

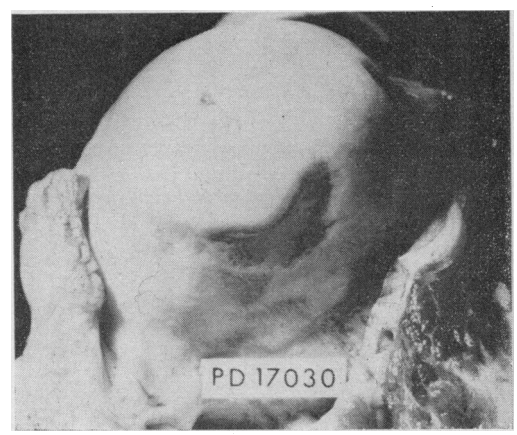

Fig 2 *Wear lesions in the humeral head*

there can be a quick turnover. For the last twenty-five years, or 15 generations, therefore, pig breeding has been intensively channelled towards producing a pig with the right carcase characteristics which grows rapidly and converts food efficiently. Little attention has been paid to the pigs' mobility.

Three types of lesion occur in the articular cartilage, and these may be called 'wear lesions', 'proliferation lesions' and 'lift lesions'. Wear lesions occur principally and earliest on the femoral and humeral heads (Figs 1 and 2). In the early lesion the cartilage in affected areas becomes thin but, until it is worn through and eburnation occurs, there is no reaction in the underlying bone. Proliferation lesions occur mostly on the humeral head and distal femur (Fig 3), and in this case there is collapse of the underlying bone and the formation of 'brood capsules' in the cartilage. Lift lesions (Fig 4) occur principally in the ulnar notch, and here the cartilage becomes detached from the bone, the primary fault probably occurring in the ossifying calcified cartilage just beneath the articular surface.

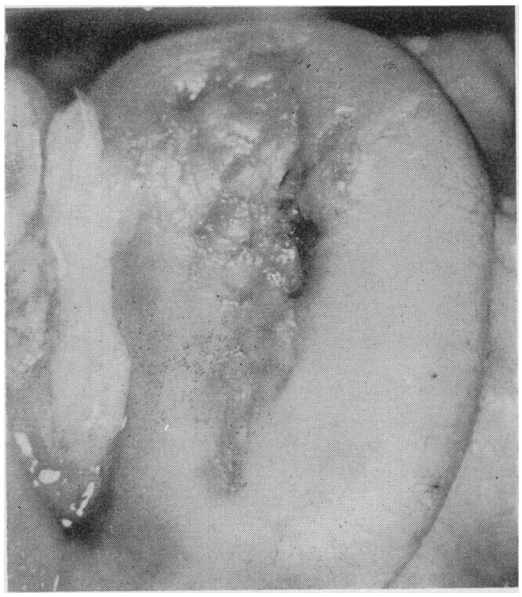

Fig 3 *Proliferation lesions on medial condyle of femur*

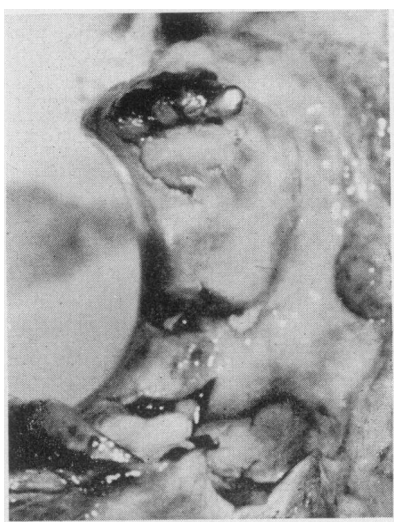

Fig 4 *Lift lesions in the ulnar notch*

The ætiology of these lesions most probably lies in the rapid growth rate and carcase characteristics that have been two of the three goals in modern pig breeding. It can be shown that bone growth in a rapidly growing pig is unable to keep pace with the growth in body weight. A similar phenomenon has been observed with respect to muscle fibre diameter (Staun 1963). Thus the rapidly grown pig of, say, 100 kg may have had less bone and thinner muscle fibres than a litter mate that has taken more time to reach the same weight. The small amount of exercise allowed to the intensively reared pig further limits muscle hypertrophy.

Schilling (1963) has made a detailed study of the effects of breeding pigs with longer backs and a greater mass of muscle on the inner aspect of the thigh. In his opinion, changes in the angles of attachment of muscles and changes in the angles of joints to accommodate greater masses of muscle contribute to the production of arthropathy.

It seems, therefore, that a number of factors predispose to joint lesions. Compared with its ancestor of twenty years ago, the modern pig has a greater body weight for its age and more muscle, although the latter may be rather weak and attached at unnatural angles to immaturely sized bones aligned in a suboptimal way. In the more cleanly conditions of some modern pig buildings, the floor is also often slippery. Under these conditions a high incidence of arthritic lesions becomes understandable.

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Traumatic Arthritis in Young Thoroughbreds

Clinically, traumatic arthritis differs fundamentally from the degenerative arthritis recorded in the mature horse and described under various names in the literature, in that it appears in the immature animal (Hare 1927, Mitchell 1937, Kelser & Callender 1938). It is considered by many to be essentially a second category of degenerative arthritis (osteoarthritis) differing from the disease of the mature animal only in its age incidence, its more rapid development and its predilection for certain joints. However, as it is almost certainly due to trauma that the first clinical signs become apparent, and as the full pathological picture is not yet established, whereas that of the older animal is essentially a non-inflammatory, degenerative disease, I prefer at the present to retain the term traumatic arthritis.

Incidence

The horse may be said to reach full skeletal maturity at about 4½ years. This disease is usually first recognized clinically in the yearling and 2-year-old thoroughbred, although occasionally it